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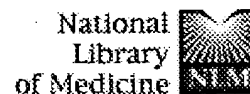
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





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
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
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
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
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
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
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
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






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
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
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
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
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
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
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
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
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






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







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
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
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
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
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











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
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
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
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
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
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
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
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
















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
















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
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
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
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
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











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
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
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
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
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
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
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
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
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
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
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
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
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






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
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
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
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
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=> s truncated apoE  
43 FILES SEARCHED...  
L1 98 TRUNCATED APOE

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AN 10333227 IFIPAT;IFIUDB;IFICDB  
TI METHODS OF SUPPRESSING MICROGLIAL ACTIVATION AND SYSTEMIC INFLAMMATORY  
RESPONSES  
IN Laskowitz Daniel T; Matthew William D; McMillian Michael  
PA Unassigned Or Assigned To Individual (68000)

RLI	US 1999-260430	19990301 CONTINUATION-IN-PART	ABANDONED
	US 2001-957909	20010921 CONTINUATION-IN-PART	PENDING
PRAI	US 1998-77551P	19980311 (Provisional)	
FI	US 2003077641	20030424	
DT	Utility; Patent Application - First Publication		
FS	CHEMICAL APPLICATION		
CLMN	80		
GI	21 Figure(s).		

FIG. 1 graphs the production of nitrite by cultures of glial cells from ApoE-deficient mice (solid bar), ApoE3 transgenic mice (hatched bar), and control mice (white bar), after exposure to lipopolysaccharide (LPS). Responses were measured at 24 and 60 hours after stimulation of cell cultures by LPS.

FIG. 2 graphs nitrite production by enriched microglia primary cultures from ApoE-deficient mice after stimulation with LPS and subsequent addition of peptides of SEQ ID NO:3 (tandem repeat peptides). Peptides were added in doses of from 0  $\mu$ M to 1000  $\mu$ M, and a dose dependent decrease in nitrite production was observed. As a control, peptides of SEQ ID NO:2 were added to cultures (solid bar); no decrease in nitrite production was observed.

FIG. 3A graphs intracellular calcium content over time in murine peritoneal macrophages, after exposure to either ApoE3 (squares) or ApoE4 (circles).

FIG. 3B graphs inositol trisphosphate (IP3) in murine peritoneal macrophages exposed to either ApoE3 (squares) or ApoE4 (circles). The graph shows the percent change in IP3 content in treated cells compared to control cells exposed to vehicle but not ApoE.

FIG. 4 graphs production of TNF alpha (picogram/ml) by microglia primary cultures from ApoE-deficient mice after addition of peptides of SEQ ID NO:6 (squares), or addition of peptides of SEQ ID NO:6 and LPS (100 ng/ml) (circles). Peptides were added in doses of 10  $\mu$ M, 100  $\mu$ M and 1000  $\mu$ M.

FIG. 5 is a graph of the optical density of cell cultures, as a measure of cell viability. Cultures of microglia from ApoEdeficient mice were exposed to either peptides of SEQ ID NO:6 (squares), or peptides of SEQ ID NO:6 and LPS (100 ng/ml) (circles). Peptides were added in doses of 10  $\mu$ M, 100  $\mu$ M and 1000  $\mu$ M.

FIG. 6 graphs production of TNF alpha (picogram/ml) by microglia primary cultures from ApoE-deficient mice after addition of peptides of SEQ ID NO:6 (squares), or addition of peptides of SEQ ID NO:6 and LPS (100 ng/ml) (circles). Peptides were added in doses of 1  $\mu$ M, 10  $\mu$ M, 100  $\mu$ M and 1000  $\mu$ M.

FIG. 7 is a graph of the optical density of cell cultures, as a measure of cell viability. Cultures of microglia from ApoEdeficient mice were exposed to either peptides of SEQ ID NO:6 (squares), or peptides of SEQ ID NO:6 and LPS (100 ng/ml) (circles). Peptides were added in doses of 10  $\mu$ M, 100  $\mu$ M and 1000  $\mu$ M.

FIG. 8. Changes in  $(Ca^{2+})_i$  in macrophages treated with apoE. Panel A: Changes in  $(Ca^{2+})_i$  in a single Fura-2/AM loaded peritoneal macrophage on stimulation with apoE (100  $\mu$ M). Details for measuring  $(Ca^{2+})_i$  are described in the Examples below. The graph shown is representative of 5 individual experiments using 20-30 cells each. Approximately 70-80% of the macrophage demonstrated changes in  $(Ca^{2+})_i$  upon stimulation with apoE. The arrow indicates the time of addition of apoE. Panel B: Effect of apoE concentration on changes in  $(Ca^{2+})_i$ . The changes in  $(Ca^{2+})_i$  in individual cells were measured prior to and following exposure to varying concentrations of apoE. The data are displayed as mean (S.E.) and are representative of two independent experiments; in each case 25-30 cells were analyzed cells per study.

FIG. 9. Changes in IP3 in macrophages treated with apoE. Panel A: Effect of apoE on IP3 synthesis in macrophages, and modulation by pertussis toxin. These results are representative of two independent experiments performed in duplicate and expressed as % change in IP3 formation at different time periods in myo-(2-3H)inositol-labeled cells stimulated with apoE (100  $\mu$ M) in the presence (open circles) and absence (filled circles) of pertussis toxin. Panel B: Effect of apoE concentration on IP3 formation in (3H)labeled macrophages. The cells were stimulated with varying concentrations of apoE for 60s and IP3 determined. Results are displayed as mean (S.E.) and are representative of two individual experiments performed in duplicate.

FIG. 10A shows the performance of mice with and without treatment on rotorodlatency after closed head injury.

FIG. 10B shows the weight gain of mice with and without treatment after



water maze latency test after closed head injury.  
 FIG. 10D shows the survival of mice with and without treatment after closed head injury.  
 FIG. 11 shows the dose-response effect of human recombinant ApoE3 on NMDA-induced cell damage. Values=mean+s.d., N=6 culture wells per condition. \*=P less-than 0.05 compared to NMDA without ApoE3. Details for measuring NMDA-induced cell damage are provided in Examples below.  
 FIG. 12(A) is a schematic of full-length ApoE and ApoE-mimetic peptides. ApoE is represented by the open box. The 10-kDa lipidbinding domain is located at the carboxyl terminus and is denoted by the shaded region. The approximate region corresponding to the ApoE LDL receptor-binding domain is depicted by the solid box (amino acids 130-150, SEQ ID NO:13), followed by the sequences of the three \*\*\*truncated\*\*\* \*\*\*apoE\*\*\*-mimetic peptides used in these studies (SEQ ID NO: 10-12). (B) Circular dichroism spectra of ApoE peptides were recorded on an Aviv Model 202 CD spectrometer, using 0.1 cm pathlength cuvettes. CD spectra of the three peptides were consistent with a mixture of helical and random coil structure.  
 FIG. 13 contains graphs showing the dose response effect of ApoE peptide (133-149) (SEQ ID NO:10) on NMDA-induced cell damage of primary mixed neuronal-glial cultures at (A) 100 mu M NMDA or (B) 300 mu M NMDA. Values=mean+s.d., N=6-8 culture wells per condition. =P less-than 0.05 compared to NMDA without peptide.  
 FIG. 14 is a graph showing the effect of truncations of ApoE peptide on NMDA-induced cell damage. Values=mean+s.d., N=8 culture wells per condition. \*=P less-than 0.05 compared to NMDA without peptide.  
 FIG. 15 is a graph showing the effect of ApoE peptide (133-149) on NMDA-induced Ca++ uptake by primary mixed neuronal-glial cultures. Values=mean+s.d., N=8 culture wells per condition. \*=P less-than 0.05 compared to NMDA without peptide.  
 FIG. 16 is a graph showing the effect of timing of ApoE peptide (133-149) exposure on NMDA-induced cell damage. Cultures were pre-treated (peptide added 24 h prior to and removed immediately before NMDA exposure), treated concurrently (peptide added immediately prior to and removed immediately after NMDA exposure), or post-treated (peptide added immediately after NMDA exposure and maintained in the medium until determination of LDH released from damaged cells 24 h later) with 6 mu M apoE peptide (133-149). Values=mean+s.d., N=8 culture wells per condition. \*=P less-than 0.05 compared to NMDA without peptide.  
 FIG. 17 contains graphs depicting the suppression of LPS-induced serum levels of TNF alpha (A) and IL-6 (B) by ApoE peptide (133149). The dark bars represent vehicle-treated animals and the light bars represent peptide-treated animals.

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 AN 2003-457197 [43] WPIDS  
 CR 1999-551213 [46]  
 DNN N2003-363657 DNC C2003-121578  
 TI Use of an ApoE peptide for manufacture of medicament for reducing neuronal cell injury associated with glutamate excitotoxicity, alleviating or reducing chronic pain, correcting or preventing substance abuse.  
 DC B04 P31  
 IN LASKOWITZ, D T; MATTHEW, W D; MCMILLIAN, M  
 PA (COGN-N) COGNOSCI INC  
 CYC 101  
 PI WO 2003026479 A2 20030403 (200343)\* EN 101p A61B000-00  
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 ZM ZW  
 ADT WO 2003026479 A2 WO 2002-US29824 20020923  
 PRAI US 2001-957909 20010921  
 IC ICM A61B000-00

L2 ANSWER 3 OF 27 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN  
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 AN 2003:526296 BIOSIS  
 DN PREV200300529419  
 TI Domains of apoE required for binding to apoE receptor 2 and to phospholipids: Implications for the functions of apoE in the brain.  
 AU Li. Xiaobing; Kvnreos. Kvriakos; Zanni. Fleni F. Zannis Vassilis

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 SO Biochemistry, (September 9 2003) Vol. 42, No. 35, pp. 10406-10417. print.  
 ISSN: 0006-2960 (ISSN print).  
 DT Article  
 LA English  
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 AN 2003:499827 BIOSIS  
 DN PREV200300501861  
 TI Molecular mechanisms of type III hyperlipoproteinemia: The contribution of  
 the carboxy-terminal domain of ApoE can account for the dyslipidemia that  
 is associated with the E2/E2 phenotype.  
 AU Kypreos, Kyriakos E.; Li, Xiaoping; van Dijk, Ko Willems; Havekes, Louis  
 M.; Zannis, Vassilis I. [Reprint Author]  
 CS Molecular Genetics, Department of Medicine, Boston University School of  
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 vzannis@bu.edu  
 SO Biochemistry, (August 26 2003) Vol. 42, No. 33, pp. 9841-9853. print.  
 ISSN: 0006-2960 (ISSN print).  
 DT Article  
 LA English  
 ED Entered STN: 29 Oct 2003  
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 DUPLICATE 4  
 AN 2003:161412 BIOSIS  
 DN PREV200300161412  
 TI Hyperlipidemia in APOE2 transgenic mice is ameliorated by a  
 \*\*\*\*truncated\*\*\*\* \*\*\*\*apoE\*\*\*\* variant lacking the C-terminal domain.  
 AU Gerritsen, Gery; Kypreos, Kyriakos E.; van der Zee, Andre; Teusink, Bas;  
 Zannis, Vassilis I.; Havekes, Louis M.; van Dijk, Ko Willems [Reprint  
 Author]  
 CS Department of Human Genetics, Leiden University Medical Center, Leiden,  
 Netherlands  
 kowvd@lumc.nl  
 SO Journal of Lipid Research, (February 2003) Vol. 44, No. 2, pp. 408-414.  
 print.  
 CODEN: JLPRAW. ISSN: 0022-2275.  
 DT Article  
 LA English  
 ED Entered STN: 26 Mar 2003  
 Last Updated on STN: 26 Mar 2003

L2 ANSWER 6 OF 27 BIOTECHDS COPYRIGHT 2004 THOMSON DERWENT/ISI on STN  
 AN 2002-17953 BIOTECHDS  
 TI Inhibiting neurofibrillary tangles formation, useful for treating e.g.  
 Alzheimer's, coronary artery disease or stroke, by reducing the formation  
 of carboxyl-terminal truncated form of apolipoprotein E in a neuron of  
 the individual;  
 neurofibrillary tangle formation inhibition and transgenic animal  
 model use in disease therapy  
 AU HUANG Y; MAHLEY R W  
 PA GLADSTONE INST J DAVID  
 PI WO 2002038108 16 May 2002  
 AI WO 2000-US51172 3 Nov 2000  
 PRAI US 2000-245737 3 Nov 2000  
 DT Patent  
 LA English  
 OS WPI: 2002-490051 [52]

L2 ANSWER 7 OF 27 IFIPAT COPYRIGHT 2004 IFI on STN DUPLICATE 6  
 AN 10204292 IFIPAT;IFIUDB;IFICDB  
 TI METHODS OF TREATING DISORDERS RELATED TO APOE  
 IN Huang Yadong; Mahley Robert W  
 PA Unassigned Or Assigned To Individual (68000)  
 PI US 2002147999 A1 20021010  
 AI US 2001-33526 20011102  
 PRAI US 2000-245737P 20001103 (Provisional)  
 FI US 2002147999 20021010

APPLICATION  
 CLMN 31  
 GI 9 Figure(s).

FIG. 1 is a graph depicting the effect of A beta 1-42 on formation of intracellular inclusions in Neuro-2A cells producing apoE.

FIG. 2 is a graph depicting the percentage of GFP-apoE3(Delta 272-299)-transfected and GFP-apoE4(Delta 272-299)-transfected cells that contain neurofibrillary tangles.

FIGS. 3A-C depict the structure of apoE (FIG. 3A); results indicating amino acids of apoE that interact with p-tau and pNF-H (FIG. 3B); and results indicating the role of the amino terminal domain of apoE in formation of neurofibrillary tangles (FIG. 3C).

FIG. 4 depicts protein blots of brain lysates from normal individuals (N1 and N2) and individuals with Alzheimer's disease (AD1, AD2, and AD3), immunoprecipitated with anti-apoE antibody.

FIG. 5 is a graph depicting carboxyl-terminal truncated apoE3 and apoE4 in brains of NSE-apoE3 and NSE-apoE4 mice.

FIG. 6 is a graph depicting age-dependent accumulation of p-tau in brains of NSE-apoE mice.

FIG. 7 is a graph depicting the occurrence of p-tau-positive intraneuronal inclusions in the hippocampus of NSE-apoE4 transgenic mice.

FIG. 8 is a graph depicting the effect of various agents on the enzymatic activity of an enzyme that catalyzes proteolytic cleavage of apoE.

FIG. 9 is a graph depicting the results of inhibition of an apoE cleaving enzyme by various peptides.

L2 ANSWER 8 OF 27 IFIPAT COPYRIGHT 2004 IFI on STN DUPLICATE 7  
 AN 10179396 IFIPAT;IFIUDB;IFICDB  
 TI COMPOUNDS AND METHODS FOR LOWERING CHOLESTEROL LEVELS WITHOUT INDUCING  
 HYPERTRIGLYCERIDEMIA; NUCLEOTIDE SEQUENCES CODING PREFERENTIAL  
 POLYPEPTIDE FOR USE IN TREATMENT OF  
 IN Kypreos Kyriakos E (GR); Zannis Vassilis I  
 PA Unassigned Or Assigned To Individual (68000)  
 PI US 2002123093 A1 20020905  
 AI US 2001-827854 20010405  
 RLI US 2000-544386 20000406 CONTINUATION-IN-PART ABANDONED  
 US 2000-679088 20001004 CONTINUATION-IN-PART PENDING  
 FI US 2002123093 20020905  
 DT Utility; Patent Application - First Publication  
 FS CHEMICAL  
 APPLICATION  
 OS CA 137:212027  
 CLMN 49  
 GI 19 Figure(s).

FIG. 1 is a schematic representation of the steps leading to the production of the apoE4-202 plasmids of the invention. The same method was used for generation of the corresponding apoE4-185, apoE4-229, and apoE4-259 plasmids.

FIGS. 2A and 2B are pictures of protein gels which demonstrate the ability of apoE4 and the truncated apoE4-202 to associate with lipoproteins in the density=1.04 to 1.21 g/ml fractions. FIGS. 2C and 2D are pictures of protein gels which demonstrate the ability of apoE4 and apoE4-202 to associate with VLDL particles.

FIG. 3A is a bar graph showing that recombinant adenoviruses expressing apoE4 increase the triglyceride levels in apoE-deficient mice. In contrast, a recombinant adenovirus expressing apoE4-202 does not cause this increase. FIG. 3B is a bar graph showing that recombinant adenoviruses expressing apoE4-202 produce a greater decrease in cholesterol levels in apoE-deficient mice than the corresponding viruses expressing full length apoE.

FIG. 4A is a bar graph and FIG. 4B is a picture of a gel demonstrating that recombinant adenoviruses expressing apoE4 or apoE4-202 synthesize comparable amounts of mRNA.

FIGS. 5A-5D are graphs showing that apoE-deficient mice infected with AdGFP-E4-202 had lower cholesterol levels on days 5 and 8 after infection than those infected with AdGFP-E4 or the AdGFP control where most of the cholesterol was found in the VLDL region. FIGS. 5E and 5F are graphs showing the increase in VLDL triglyceride levels in mice on days 5 and 8 after infection with AdGFP-E4. In contrast, infection with AdGFP-E4-202 did no increase triglyceride levels.

FIG. 6 is a graph showing that the plasma of apoE-deficient mice infected with AdGFP-E4 or AdGFP-E4-202 have similar levels of lipid-free apoE protein (fractions 22-25). In the plasma of mice infected with AdGFP-E4, approximately 50% of the total apoE is distributed in the HDL fractions

mice infected with AdGFP-E4-202, the \*\*\*\*truncated\*\*\* \*\*\*apoE\*\*\* protein is present at a lower level than the full length apoE in mice infected with AdGFP-E4 and is uniformly distributed in all lipoprotein fractions.

FIG. 7A is a picture of a protein gel illustrating that in apoE-deficient mice infected with the recombinant adenovirus expressing apoE4, the apoE4 displaces other proteins from the VLDL density particles. No displacement of other proteins from the VLDL density particles is detected in mice infected with recombinant adenovirus expressing apoE4-202. FIG. 7B is a schematic illustration showing how the displacement of other proteins from the VLDL density particles by apoE4 could be the cause of hypertriglyceridemia. FIG. 7C is a schematic illustration of the putative domains of apoE.

FIGS. 8A and 8B are bar graphs showing an in vivo time course analysis of serum triglyceride (FIG. 8A) and cholesterol levels (FIG. 8B) in apoE-deficient mice infected with an AdGFP control (no apoE), AdGFP-E4, AdGFP-E4-229, or AdGFP-E4-259. Infection of apoE-deficient mice with a dose of  $4 \times 10^9$  pfu of the adenoviruses expressing the truncated E4-229 or E4-259 forms significantly reduced the level of cholesterol without increasing the level of serum triglycerides. In contrast, infection with a dose of  $2 \times 10^9$  pfu of the adenovirus expressing wild-type E4 does not appear to reduce cholesterol and causes a dramatic increase in serum triglyceride levels. The control virus AdGFP does not appear to have any effects on the basal (day 0) cholesterol and triglyceride levels of the apoE-deficient mice.

FIGS. 9A-9D are graphs showing the cholesterol FPLC lipoprotein profiles of serum samples from apoE-deficient mice infected with  $2 \times 10^9$  pfu of AdGFP-E4 or  $4 \times 10^9$  pfu of AdGFP control virus (FIGS. 9A and 9B) or with  $4 \times 10^9$  pfu of AdGFP-E4-229 or AdGFP-E4-259 (FIGS. 9C and 9D). On days five and eight after infection, serum samples were collected and fractionated by FPLC on a Sepharose 6 column. Fractions were then analyzed for cholesterol content.

FIGS. 10A-10D are graphs showing triglyceride FPLC lipoprotein profiles of serum samples from apoE-deficient mice infected with  $2 \times 10^9$  pfu of AdGFP-E4 or  $4 \times 10^9$  pfu of AdGFP control virus (FIG. 10A and 11B) or with  $4 \times 10^9$  pfu of AdGFP-E4-229 or AdGFP-E4-259 (FIG. 10C and 10D). On days five and eight after infection, serum samples were collected and fractionated by FPLC on a Sepharose 6 column. Fractions were then analyzed for triglyceride content.

FIGS. 11A and 11B are a Northern blot and a bar graph showing the in vivo mRNA expression of wt-apoE4, apoE4-229 and apoE4-259. ApoE-deficient mice infected with the indicated doses of the AdGFP-E4, AdGFP-E4-229 or AdGFP-E4-259 viruses were sacrificed five days after infection, and liver samples were collected. Then total RNA from the liver samples was isolated and analyzed by Northern blot analysis for the expression of apoE and GAPDH; a representative autoradiogram is shown in FIG. 11A. The apoE mRNA levels normalized for GAPDH mRNA levels are graphed in FIG. 11B, showing that all three apoE mRNAs are expressed to similar levels. ApoE4 causes hypertriglyceridemia and fails to clear cholesterol; in contrast, apoE4-229 and apoE4-259 drastically reduce cholesterol without the unwanted side-effect of hypertriglyceridemia (FIGS. 11C and 11D).

FIG. 12 is a bar graph of the average rate of VLDL triglyceride production in vivo for apoE-deficient mice infected with AdGFP-E4-259, AdGFP-E4, or control AdGFP virus. Wild-type apoE4 induces a dramatic increase in VLDL triglyceride production compared to that induced by apoE4-259 or control virus. This failure of apoE4-259 to induce VLDL triglyceride production may contribute to the inability of the \*\*\*\*truncated\*\*\* \*\*\*apoE\*\*\* mutants to trigger hypertriglyceridemia.

FIGS. 13A-13C are pictures of gels showing that apoE4-229 and apoE4-259 can associate with smaller (higher density) apoE-deficient VLDL particles in addition to associating with the large (lowest density) apoE-deficient VLDL particles. The numbers below each lane of the gels represent the amount of apoE4 present in each lane in mg/dl, as determined by ELISA. The degree of association of apoE to apoE-deficient VLDL is similar for wild-type apoE4 and the apoE4-229 and apoE4-259 truncated forms. The truncated forms of apoE4 have a greater association than wild-type apoE for higher density VLDL particles, which are smaller in size and have a lower triglyceride composition.

FIG. 14 is a schematic diagram of a model of the effects of overexpression of wild-type apoE, apoE-229, or apoE-259 on VLDL and chylomicron catabolism in vivo.

FIG. 15 is a picture of the SDS-PAGE analysis of the culture medium of HTB-13 cells infected with adenoviruses expressing apoE3, apoE4, apoE4-202 or apoE4-185. Fifteen  $\mu$ l of culture medium were analyzed.

levels, respectively of apoE-deficient and C57BL6 mice infected with either the control adenovirus AdGFP, a recombinant adenoviruses expressing wild-type apoE, or a recombinant adenoviruses expressing a \*\*\*truncated\*\*\* \*\*\*apoE\*\*\*. Mice were infected in triplicate with the indicated doses of the indicated recombinant adenovirus, and serum samples were isolated and analyzed for cholesterol and triglyceride levels on the indicated days after infection as described herein. FIG. 17A is a picture of a representative autoradiograms of Northern blot analysis of mice infected with the indicated dose of the control adenoviruses AdGFP, a recombinant adenoviruses expressing wild-type apoE, or a recombinant adenoviruses expressing a \*\*\*truncated\*\*\* \*\*\*apoE\*\*\*. Total RNA was isolated from the livers of the infected mice on the indicated days after infection and analyzed by Northern blotting for the expression of apoE mRNA. FIG. 17A also shows the ethidium bromide staining of the gel for 18S ribosomal RNA as a control of RNA loading and integrity. FIG. 17B is a bar graph showing triglyceride levels of the individual mice expressed in mg/dl. FIG. 17B is a bar graph showing cholesterol levels of the individual mice expressed in mg/dl. FIGS. 18A-18D are graphs of the FPLC profiles of cholesterol and triglycerides of mice infected with the apoE4-185 (FIGS. 18A and 18C, respectively) or apoE4 (FIGS. 18B and 18D, respectively) expressing adenovirus. Five days after infection of mice with either 2 x 10<sup>9</sup> pfu of the recombinant adenovirus expressing AdGFP-E4 or 1 x 10<sup>10</sup> pfu of the recombinant adenovirus expressing AdGFP-E4-185, serum samples were obtained. These serum samples were fractionated by FPLC, and the cholesterol and triglyceride levels of each FPLC fraction were determined as described herein. FIG. 19 is a bar graph of the average rate of hepatic VLDLtriglyceride production analysis in mice infected with AdGFP, AdGFP-E4, or AdGFP-E4-185. The bar-graph represents the mean+/standard deviation of the individual rates of VLDL-triglyceride production per virus group.

L2 ANSWER 9 OF 27 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN  
 DUPLICATE 8  
 AN 2002:390964 BIOSIS  
 DN PREV200200390964  
 TI \*\*\*Truncated\*\*\* \*\*\*apoE\*\*\* forms tangle-like structures in a neuronal cell line.  
 AU Ljungberg, M. Cecilia; Dayanandan, Rejith; Asuni, Ayodeji; Rupniak, Tom H.; Anderton, Brian H.; Lovestone, Simon [Reprint author]  
 CS Old Age Psychiatry, Institute of Psychiatry, King's College London, De Crespigny Park, London, SE5 8AF, UK  
 SO Neuroreport, (7 May, 2002) Vol. 13, No. 6, pp. 867-870. print.  
 CODEN: NERPEZ. ISSN: 0959-4965.  
 DT Article  
 LA English  
 ED Entered STN: 17 Jul 2002  
 Last Updated on STN: 17 Jul 2002

L2 ANSWER 10 OF 27 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN  
 DUPLICATE 9  
 AN 2001:403263 BIOSIS  
 DN PREV200100403263  
 TI Apolipoprotein E fragments present in Alzheimer's disease brains induce neurofibrillary tangle-like intracellular inclusions in neurons.  
 AU Huang, Yadong [Reprint author]; Liu, Xiao Qin; Wyss-Coray, Tony; Brecht, Walter J.; Sanan, David A.; Mahley, Robert W.  
 CS Gladstone Institute of Neurological Disease, San Francisco, CA, 94141-9100, USA  
 yhuang@gladstone.ucsf.edu  
 SO Proceedings of the National Academy of Sciences of the United States of America, (July 17, 2001) Vol. 98, No. 15, pp. 8838-8843. print.  
 CODEN: PNASA6. ISSN: 0027-8424.  
 DT Article  
 LA English  
 ED Entered STN: 22 Aug 2001  
 Last Updated on STN: 22 Feb 2002

L2 ANSWER 11 OF 27 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN  
 DUPLICATE 10  
 AN 2001:295780 BIOSIS  
 DN PREV200100295780  
 TI The amino-terminal 1-185 domain of apoE promotes the clearance of lipoprotein remnants in vivo. The carboxy-terminal domain is required for

M.; Zannis, Vassilis I. [Reprint author]  
 CS Department of Medicine, Whitaker Cardiovascular Institute, Boston  
 University School of Medicine, 715 Albany St., W509, Boston, MA,  
 02118-2394, USA  
 vzannis@bu.edu  
 SO Biochemistry, (May 22, 2001) Vol. 40, No. 20, pp. 6027-6035. print.  
 CODEN: BICHAW. ISSN: 0006-2960.  
 DT Article  
 LA English  
 ED Entered STN: 20 Jun 2001  
 Last Updated on STN: 19 Feb 2002

L2 ANSWER 12 OF 27 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN  
 DUPLICATE 11  
 AN 2001:574182 BIOSIS  
 DN PREV200100574182  
 TI Vascular-derived thrombin generates neurotoxic apolipoprotein E fragments.  
 AU Grammas, P. [Reprint author]; Ottman, T. [Reprint author];  
 Reimann-Philipp, U. [Reprint author]  
 CS Oklahoma Center of Neuroscience, University of Oklahoma HSC, Oklahoma  
 City, OK, USA  
 SO Society for Neuroscience Abstracts, (2001) Vol. 27, No. 2, pp. 2002.  
 print.  
 Meeting Info.: 31st Annual Meeting of the Society for Neuroscience. San  
 Diego, California, USA. November 10-15, 2001.  
 ISSN: 0190-5295.  
 DT Conference; (Meeting)  
 Conference; Abstract; (Meeting Abstract)  
 LA English  
 ED Entered STN: 12 Dec 2001  
 Last Updated on STN: 25 Feb 2002

L2 ANSWER 13 OF 27 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN  
 AN 2001:935954 SCISEARCH  
 GA The Genuine Article (R) Number: 487UW  
 TI Hyperlipidemia in APOE2 transgenic mice is aggravated by overexpression of  
 full length APOE3 whereas it is reduced by a \*\*\*truncated\*\*\*  
 \*\*\*ApoE\*\*\* variant  
 AU Gerritsen G (Reprint); Kypreos K E; Van der Zee A; Zannis V I; Havekes L  
 M; van Dijk K W  
 CS Leiden Univ, Med Ctr, Leiden, Netherlands; Boston Univ, Sch Med, Boston,  
 MA 02118 USA; TNO, PG, Leiden, Netherlands  
 CYA Netherlands; USA  
 SO CIRCULATION, (23 OCT 2001) Vol. 104, No. 17, Supp. [S], pp. 114-115. MA  
 552.  
 Publisher: LIPPINCOTT WILLIAMS & WILKINS, 530 WALNUT ST, PHILADELPHIA, PA  
 19106-3621 USA.  
 ISSN: 0009-7322.  
 DT Conference; Journal  
 LA English  
 REC Reference Count: 0

L2 ANSWER 14 OF 27 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN  
 AN 2002:263588 BIOSIS  
 DN PREV200200263588  
 TI Hyperlipidemia in APOE2 transgenic mice is aggravated by overexpression of  
 full length APOE3 whereas it is reduced by a \*\*\*truncated\*\*\*  
 \*\*\*ApoE\*\*\* variant.  
 AU Gerritsen, Gery [Reprint author]; Kypreos, Kyriakos E.; van der Zee,  
 Andre; Zannis, Vassilis I.; Havekes, Louis M.; van Dijk, Ko Willems  
 CS Leiden Univ Med Ctr, Leiden, Netherlands  
 SO Circulation, (October 23, 2001) Vol. 104, No. 17 Supplement, pp.  
 II.114-II.115. print.  
 Meeting Info.: Scientific Sessions 2001 of the American Heart Association.  
 Anaheim, California, USA. November 11-14, 2001. American Heart  
 Association.  
 CODEN: CIRCAZ. ISSN: 0009-7322.  
 DT Conference; (Meeting)  
 Conference; Abstract; (Meeting Abstract)  
 LA English  
 ED Entered STN: 1 May 2002  
 Last Updated on STN: 1 May 2002

L2 ANSWER 15 OF 27 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN

DN PREV200000369084  
TI Structural determinants in the C-terminal domain of apolipoprotein E  
mediating binding to the protein core of human aortic biglycan.  
AU Klezovitch, Olga [Reprint author]; Formato, Marilena; Cherchi, Gian M.;  
Weisgraber, Karl H.; Scanu, Angelo M.  
CS Dept. of Medicine, University of Chicago, 5841 S. Maryland Ave., Chicago,  
IL, 60637, USA  
SO Journal of Biological Chemistry, (June 23, 2000) Vol. 275, No. 25, pp.  
18913-18918. print.  
CODEN: JBCHA3. ISSN: 0021-9258.  
DT Article  
LA English  
ED Entered STN: 30 Aug 2000  
Last Updated on STN: 8 Jan 2002

L2 ANSWER 16 OF 27 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN  
AN 2001:98285 BIOSIS  
DN PREV200100098285  
TI Increased ratio of truncated to full-length apoE in Alzheimer's brain.  
AU Zhang, D. [Reprint author]; McQuade, J. A.; Shockley, K. P.; Marques, M.  
A.; Crutcher, K. A.  
CS University of Cincinnati, Cincinnati, OH, USA  
SO Society for Neuroscience Abstracts, (2000) Vol. 26, No. 1-2, pp. Abstract  
No.-576.7. print.  
Meeting Info.: 30th Annual Meeting of the Society for Neuroscience. New  
Orleans, LA, USA. November 04-09, 2000. Society for Neuroscience.  
ISSN: 0190-5295.  
DT Conference; (Meeting)  
LA English  
ED Entered STN: 21 Feb 2001  
Last Updated on STN: 15 Feb 2002

L2 ANSWER 17 OF 27 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN  
DUPLICATE 13  
AN 1999:403496 BIOSIS  
DN PREV199900403496  
TI Truncated apolipoprotein E (ApoE) causes increased intracellular calcium  
and may mediate ApoE neurotoxicity.  
AU Tolar, Martin; Keller, Jeffrey N.; Chan, Stephen; Mattson, Mark P.;  
Marques, Marcos A.; Crutcher, Keith A. [Reprint author]  
CS Department of Neurosurgery, University of Cincinnati, Cincinnati, OH,  
45267-0515, USA  
SO Journal of Neuroscience, (Aug. 15, 1999) Vol. 19, No. 16, pp. 7100-7110.  
print.  
CODEN: JNRSDS. ISSN: 0270-6474.  
DT Article  
LA English  
ED Entered STN: 8 Oct 1999  
Last Updated on STN: 8 Oct 1999

L2 ANSWER 18 OF 27 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN  
AN 2000:141120 BIOSIS  
DN PREV200000141120  
TI \*\*\*Truncated\*\*\* \*\*\*apoE\*\*\* in human brain: Fact or artifact?.  
AU McQuade, J.-A. M. [Reprint author]; Zhang, D. S. [Reprint author];  
Crutcher, K. A. [Reprint author]  
CS Dept. of Neurosurgery, U. of Cincinnati, Cincinnati, OH, 45267-0515, USA  
SO Society for Neuroscience Abstracts, (1999) Vol. 25, No. 1-2, pp. 1345.  
print.  
Meeting Info.: 29th Annual Meeting of the Society for Neuroscience. Miami  
Beach, Florida, USA. October 23-28, 1999. Society for Neuroscience.  
ISSN: 0190-5295.  
DT Conference; (Meeting)  
LA English  
ED Entered STN: 19 Apr 2000  
Last Updated on STN: 4 Jan 2002

L2 ANSWER 19 OF 27 CAPLUS COPYRIGHT 2004 ACS on STN  
AN 1998:790244 CAPLUS  
DN 130:181089  
TI Frequency of apoB and apoE gene mutations as causes of  
hypobetalipoproteinemia in the Framingham offspring population  
AU Weltv. Francine K.; Lahoz Carlos; Tucker Katherine L.; Anderson Jace M.

SO Tufts University, Boston, MA, 02215, USA  
Arteriosclerosis, Thrombosis, and Vascular Biology (1998), 18(11),  
1745-1751

CODEN: ATVBFA; ISSN: 1079-5642

PB Lippincott Williams & Wilkins

DT Journal

LA English

RE.CNT 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 20 OF 27 CABA COPYRIGHT 2004 CABI on STN

AN 94:90243 CABA

DN 19941408093

TI Familial apolipoprotein E deficiency and type III hyperlipoproteinemia due  
to a premature stop codon in the apolipoprotein E gene

AU Lohse, P.; Brewer, B.; Meng, M. S.; Skarlatos, S. I.; LaRosa, J. C.;  
Brewer, H. B., Jr.

CS Bldg 10, Room 7N117, National Institute of Health, 9000 Rockville Pike,  
Bethesda, MD 20892, USA.

SO Journal of Lipid Research, (1992) Vol. 33, No. 11, pp. 1583-1590. 42 ref.  
ISSN: 0022-2275

DT Journal

LA English

ED Entered STN: 19941101

Last Updated on STN: 19941101

L2 ANSWER 21 OF 27 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN  
DUPLICATE 14

AN 1989:331043 BIOSIS

DN PREV198988034043; BA88:34043

TI HUMAN APOLIPOPROTEIN E RECEPTOR BINDING ACTIVITY OF TRUNCATED VARIANTS  
WITH CARBOXYL-TERMINAL DELETIONS.

AU LALAZAR A [Reprint author]; OU S-H I; MAHLEY R W

CS GLADSTONE FOUND LAB, PO BOX 40608, SAN FRANCISCO, CALIF 94140-0680, USA

SO Journal of Biological Chemistry, (1989) Vol. 264, No. 15, pp. 8447-8450.  
CODEN: JBCHA3. ISSN: 0021-9258.

DT Article

FS BA

LA ENGLISH

ED Entered STN: 20 Jul 1989

Last Updated on STN: 20 Jul 1989

L2 ANSWER 22 OF 27 DGENE COPYRIGHT 2004 THOMSON DERWENT on STN

AN AAO18046 peptide DGENE

TI Inhibiting neurofibrillary tangles formation, useful for treating e.g.  
Alzheimer's, coronary artery disease or stroke, by reducing the formation  
of carboxyl-terminal truncated form of apolipoprotein E in a neuron of  
the individual -

IN Huang Y; Mahley R W

PA (GLAD-N) GLADSTONE INST J DAVID.

PI WO 2002038108 A2 20020516

75p

AI WO 2001-US51172 20011102

PRAI US 2000-245737P 20001103

DT Patent

LA English

OS 2002-490051 [52]

DESC C-terminal \*\*\*truncated\*\*\* \*\*\*apoE\*\*\* formation inhibitor peptide  
#4.

L2 ANSWER 23 OF 27 DGENE COPYRIGHT 2004 THOMSON DERWENT on STN

AN AAO18045 peptide DGENE

TI Inhibiting neurofibrillary tangles formation, useful for treating e.g.  
Alzheimer's, coronary artery disease or stroke, by reducing the formation  
of carboxyl-terminal truncated form of apolipoprotein E in a neuron of  
the individual -

IN Huang Y; Mahley R W

PA (GLAD-N) GLADSTONE INST J DAVID.

PI WO 2002038108 A2 20020516

75p

AI WO 2001-US51172 20011102

PRAI US 2000-245737P 20001103

DT Patent

LA English

OS 2002-490051 [52]

DESC C-terminal \*\*\*truncated\*\*\* \*\*\*apoE\*\*\* formation inhibitor peptide



L2 ANSWER 24 OF 27 DGENE COPYRIGHT 2004 THOMSON DERWENT on STN  
 AN AAO18044 peptide DGENE  
 TI Inhibiting neurofibrillary tangles formation, useful for treating e.g. Alzheimer's, coronary artery disease or stroke, by reducing the formation of carboxyl-terminal truncated form of apolipoprotein E in a neuron of the individual -  
 IN Huang Y; Mahley R W  
 PA (GLAD-N) GLADSTONE INST J DAVID.  
 PI WO 2002038108 A2 20020516 75p  
 AI WO 2001-US51172 20011102  
 PRAI US 2000-245737P 20001103  
 DT Patent  
 LA English  
 OS 2002-490051 [52]  
 DESC C-terminal \*\*\*truncated\*\*\* \*\*\*apoE\*\*\* formation inhibitor peptide #2.

L2 ANSWER 25 OF 27 DGENE COPYRIGHT 2004 THOMSON DERWENT on STN  
 AN AAO18043 peptide DGENE  
 TI Inhibiting neurofibrillary tangles formation, useful for treating e.g. Alzheimer's, coronary artery disease or stroke, by reducing the formation of carboxyl-terminal truncated form of apolipoprotein E in a neuron of the individual -  
 IN Huang Y; Mahley R W  
 PA (GLAD-N) GLADSTONE INST J DAVID.  
 PI WO 2002038108 A2 20020516 75p  
 AI WO 2001-US51172 20011102  
 PRAI US 2000-245737P 20001103  
 DT Patent  
 LA English  
 OS 2002-490051 [52]  
 DESC C-terminal \*\*\*truncated\*\*\* \*\*\*apoE\*\*\* formation inhibitor peptide #1.

L2 ANSWER 26 OF 27 FEDRIP COPYRIGHT 2004 NTIS on STN  
 AN 2003:142140 FEDRIP  
 NR CRISP 5R01AG20249-02  
 TI Proteolysis of apoE and Alzheimer's pathology  
 SF Principal Investigator: CRUTCHER, KEITH A; CRUTCHKA@EMAIL.UC.EDU, CRUTCHER, KEITH A., PHD, 231 ALBERT SABIN WAY, ML # 0515  
 CSP UNIVERSITY OF CINCINNATI, CINCINNATI, OHIO  
 CSS Supported By: NATIONAL INSTITUTE ON AGING  
 DB 2009 (/30/02)  
 FYR 2003  
 DE 2008 (/31/07)  
 FU Noncompeting Continuation (Type 5)  
 FS National Institutes of Health

L2 ANSWER 27 OF 27 TOXCENTER COPYRIGHT 2004 ACS on STN  
 AN 2003:153136 TOXCENTER  
 DN CRISP-2002-AG20249-01A1  
 TI Proteolysis of apoE and Alzheimer's pathology  
 AU CRUTCHER K A  
 CS CRUTCHKA@EMAIL.UC.EDU, CRUTCHER, KEITH A., PHD, 231 ALBERT SABIN WAY, CINCINNATI, OH 45267-0515:OHIO  
 CSS U.S. DEPT. OF HEALTH AND HUMAN SERVICES; PUBLIC HEALTH SERVICE; NATIONAL INSTITUTES OF HEALTH, NATIONAL INSTITUTE ON AGING  
 SO Crisp Data Base National Institutes of Health.  
 DT (Research)  
 FS CRISP  
 LA English  
 ED Entered STN: 20030708  
 Last Updated on STN: 20030708  
 STN INTERNATIONAL LOGOFF AT 10:39:28 ON 02 JAN 2004